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Editorials

THE WORKING OF THE NEW TUBERCULOSIS ORDER.

THE working of the new Tuberculosis Order has started well, and in all parts of the country the veterinary inspectors have found much work to do. Its justice is universally acknowledged, and the benefit to human beings by the closing down of the source which gave tuberculosis to thousands of helpless children will with certainty be manifested in the medical statistics of the next few years.

THE INTRADERMAL TUBERCULIN TEST.

IN a very interesting paper on "The Outlines of Tuberculosis Eradication," by Dr. J. R. Mohler, chief of the U.S.A. Bureau of Animal Industry, and published in *Veterinary Medicine* for October 1925, it is stated that in America in 1920 there were several hundred thousand cattle tested by the subcutaneous method, but comparatively few by the intradermal method. The records show that for the first eight months of the current fiscal year, 2,788 cattle were subcutaneously tested, whereas 3,356,000 cattle were intradermally tested. It is evident, therefore, that this latter method has, in America, already forged ahead as a more convenient way of applying the tuberculin test. It is now on its trial in Great Britain, and we await with interest the opinions of practitioners as to its advantages and greater reliability.

THE DAIRY SHOW AND CLEANLINESS IN MILKING.

MOST of us begin to realise that there is a very real demand for some suitable device which will milk cows and so replace hand-milkers.

XUM

The great interest shown at the Dairy Show this year in milking machines indicates that the milk producer is keenly on the lookout for something which will replace his troublesome hand-milkers, and so render him less dependent upon labour, the rise in wages, and the incessant demand for Saturday afternoons and Sundays off.

At the same time the consumer is being urged by the authorities and encouraged by societies to demand cleaner milk.

Certified Milk T.T. is being practically universally used as a matter of course in all our large hospitals. Lord Rayleigh's Dairies, Ltd., The Kerry Estates, Ltd., Park Farm, Culverthorpe, and many others are producing large quantities of this highest grade of milk for the public, and finding it to be a paying proposition. These producers find that a milking machine is as necessary a part of their equipment as is a steriliser, because the machines eliminate the first and greatest source of infection of the milk, namely between the udder and the pail.

This raises the question as to whether the latest machines are easy to clean, and whether the infection, if any, introduced by such machines is less than that which may be introduced by the hands, together with the dust which settles down into the pail during the process of hand-milking.

It is evident that the infection is not greater with the latest machines than with hand-milking, otherwise the producers of certified milk would be compelled to abandon their milking machines immediately.

However much we may dislike mechanical milking, we shall have to train ourselves to look upon it as a necessary evil, unless some new and greatly improved machine is introduced which once and for all by its good performance eradicates from our memory all the evil consequences of the failures in the past.

When such a new machine is discovered we shall require proof of its satisfactory working over several years before we can recommend its adoption with confidence. This will be specially the case with anyone who has experienced unsatisfactory results with some other milker.

Search must be made abroad, and particularly in such countries as Sweden, Denmark, and Holland, which are the homes of the dairy industry, and pioneers in its development.

The United States of America are always ready to try any new device which can be shown to save labour.

The ideal machine must be simple in construction, easy to clean, with a minimum of moving parts in the cowshed; and if possible there must be a total abandonment of the intricate pulsators on the pails.

Finally, and the most important, the act of milking must as nearly reproduce the action of the calf's mouth as is possible, whilst a large number of users can guarantee to us that there is no possibility of injury to the cows.

THE RETURN OF FOOT-AND-MOUTH DISEASE.

THE recent recrudescence of foot-and-mouth disease in England has taught us the necessity for constant vigilance on the part of the veterinary officials of the Ministry of Agriculture, and it is well for the country that a firm policy is still being adopted. The mysteriousness of the origin of this disease still baffles our scientists, and it is hard at this time of year to reconcile these recent outbreaks with the migration of birds. It is truly the most mysterious of all the cattle diseases with which we have to deal, and all countries of the world would crown with honour the investigator who would give us the true solution of the difficulty.

General Articles

STUDIES IN RINDERPEST IMMUNITY: (I) SUSCEPTIBILITY AND RESISTANCE.

By H. E. HORNBY, F.R.C.V.S.,
AND
G. N. HALL, M.R.C.V.S.,
From the Veterinary Pathological Laboratory, Mpapwa, Tanganyika Territory.

Note.—In these preliminary articles references to literature will not be given; only in the final article of this series will notice be taken of the good work on rinderpest which has been done, and is being done, in other laboratories. For the present we shall set down merely what we have found to occur, and the conclusions we have drawn from these occurrences, irrespective of whether they are in agreement with commonly accepted theories or not.

The cattle used in our experiments were all young bulls and oxen, hereafter referred to indifferently as "oxen," from the Iringa district of Tanganyika Territory. In that district there has not been any rinderpest for at least five years, and we find that more than 90 per cent. of the young oxen which come from there are susceptible to infection.

Our strain of rinderpest virus has been maintained in this Laboratory for more than three years. Ordinarily it is propagated by means of weekly subcutaneous inoculations, but every few months some irregularity of reaction breaks the sequence of such inoculations, and transference has then been made by contact or by intravenous

injection. Throughout these years the virus has shown no signs of altering in virulence; the results of inoculation of virus into susceptible Iringa oxen have fluctuated around the same mean during the whole period. The expected result when a sufficiently large number of such oxen are inoculated, each with 5 c.c. of fresh, virulent blood, is that all which are susceptible, i.e. between 90 per cent. and 100 per cent., will react, and of these approximately one-third will die from rinderpest, one-third will recover after good reactions which include other clinical symptoms than merely a temperature reaction, and one-third will recover after a temperature reaction only.

Charts I and II illustrate the different courses which may be taken by the disease in two apparently similar animals inoculated at the same time with an equal amount of fresh, virulent blood from the same flask. Ox 1378 was destroyed, as unlikely to live, a fortnight after inoculation, whereas Ox 1379 had by that time completely recovered from a mild, thermal only, reaction. Such difference in result is usually spoken of as due to difference in susceptibility, and one of the purposes of this article is to show that it is really due to difference in resistance after the body has been invaded.

Before going further it is necessary to define the two words *Susceptibility* and *Resistance*. Since all cattle are either susceptible to rinderpest or immune, therefore susceptibility is, when used in this sense, merely the opposite of immunity, and means that a beast is liable to contract rinderpest when exposed to infection. *Susceptibility, then, represents a condition which allows the rinderpest virus to get sufficient "foothold" within the tissues of its host to cause symptoms of disease.*

Resistance, on the other hand, goes beyond mere susceptibility. Its meaning lies between that of immunity, which is complete resistance, and susceptibility, which is absence or feebleness of resistance against invasion. It indicates not only that opposition to the virus obtaining a "foothold" which can be covered by the term lessened susceptibility, but also the whole of the fight put up by the body against the action of the virus after this has got its hold; and it is the success of this part of the resistance which decides the course of the disease. It is because we wish to emphasise an essential difference between (1) the opposition to the virus obtaining a "foothold," and (2) any subsequent opposition, that we ask that for the purpose of this article *Susceptibility* may be defined as above, while *by Resistance may be understood the rapidity and efficiency with which the defensive forces of the body are mobilised against the invading virus*, after this has already obtained a "foothold" by reason of the body's susceptibility.

In the relation of cattle to rinderpest there is no such thing as partial susceptibility; because if a beast is not susceptible it is immune, and then no exposure to infection will set up the least trace of the disease. During hyperimmunisation an immune ox receives two or more litres of virulent blood, yet in 1,500 cases where the temperatures were taken daily after the hyperimmunising doses had been given there was not a single thermal or other reaction which could not be interpreted as either surgical, anaphylactoid, or intercurrent. The solidity of rinderpest immunity is one of its most wonderful features.

When a susceptible beast is exposed to sufficient infection it will

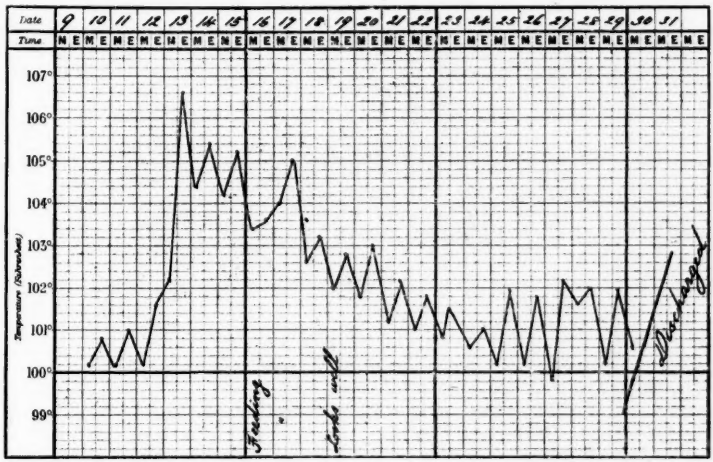

CHART I.

develop symptoms of rinderpest which may be anything between three days' slight fever, which causes no visible ill-effect, and those of acute hæmorrhagic gastro-enteritis culminating in death.

Were such vast differences in the courses which the disease may take dependent on corresponding differences in susceptibility—as the current phrases "highly susceptible" or "not very susceptible" would indicate—then in a number of Iringa oxen taken at random we should find that some were much more easily infected than others, and the *minimal infective dose* would vary accordingly, in that a small dose of virus just sufficient to infect a highly susceptible beast would fail to infect one less susceptible. On the other hand, if susceptibility be a constant, and so independent of the course run

by the disease, then the amount of virus just sufficient to set up the disease in one ox will be just sufficient to set up the disease in all, i.e. the minimal infective dose of any concentration of virus will be the same for all animals.

To test the assumption that susceptibility is a constant we carried out a series of experiments. We made a series of dilutions of virulent fluid—as blood, peritoneal washings, or liver extract—and injected a measured amount of each of these dilutions into a susceptible ox. It is obvious that if susceptibility be a constant, then the dilution below that of which the injected quantity contains a minimal infective dose will not be infective in the same dose, and

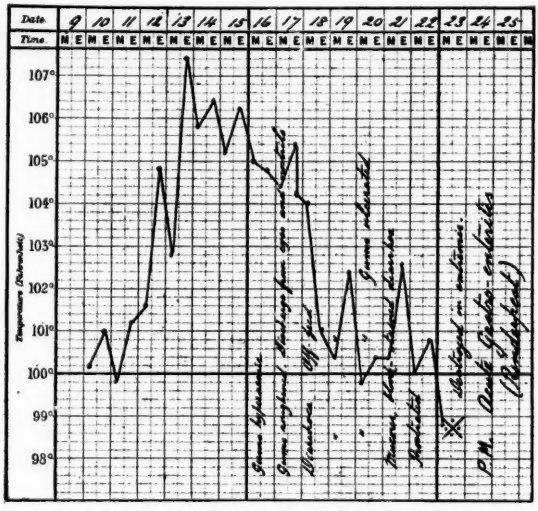
Date
9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
Time
M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M
Temperature (Fahrenheit)
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°

CHART II.

that all dilutions of greater concentration than this will infect. On the other hand, if susceptibility vary within such wide limits as does the course of the disease, then there will be no regularity in the results which follow injection.

A number of preliminary experiments, which need not be mentioned here, had taught us that a series of dilutions calculated to give useful results without wastage of experimental animals were dilutions of which 20 c.c. contained respectively 0·2 c.c., 0·02 c.c., and 0·002 c.c. of the original infective fluid. The first experiment of the series will be described in some detail; the others, being similar, need only be indicated by results.

EXPERIMENT 1. **15/8/23.** A Virus Control (Ox No. 928) commenced to react on the third day following inoculation with 20 c.c. citrated virulent blood, and was bled on the fourth day of reaction, or seventh day after inoculation. Its morning temperature on that day was 103·4° F. Some blood was drawn into one-ninth of its volume of 7½ per cent. citrate solution; 20 c.c. of this were transferred to a flask containing 160 c.c. of normal saline. From this second flask 20 c.c. were transferred to a third flask containing 180 c.c. of saline, and so on into fourth and fifth flasks. The result was that 20 c.c. of the mixture in No. 3 flask contained 0·2 c.c. of original blood, 20 c.c. of the contents of No. 4 flask contained 0·02 c.c., and 20 c.c. from No. 5 flask contained 0·002 c.c. of blood. Three supposedly susceptible oxen, in a clean shed, and attended by boys who did not come in contact with other sources of infection, were inoculated subcutaneously with this series of dilutions, each beast getting 20 c.c. of its appropriate flask's contents. After seven days, the beast which failed to react was given a test dose of 10 c.c. of virulent blood to ascertain if the non-reaction was due to immunity or to weakness of virus. No account was taken of a possible delayed reaction of more than eight days, as experience has taught us that this rarely occurs.

Ox 943 received 0·002 c.c. It did not react, though susceptible.
Ox 942 received 0·02 c.c. It reacted the fourth day.
Ox 941 received 0·2 c.c. It reacted the third day.

EXPERIMENT 2. **22/8/23.** Virus Control (Ox 939). Reacted third day. Bled seventh day. Morning temperature 102° (being low owing to debility).

Ox 948 received 0·002 c.c. It did not react. Tested: Immune.
Ox 949 received 0·02 c.c. It reacted the third day.
Ox 950 received 0·2 c.c. It reacted the fourth day.

EXPERIMENT 3. **12/9/23.** Serum Tester (Ox 966). Reacted fourth day. Bled seventh day after double inoculation with virus and serum. Morning temperature 103·6°.

Ox 972 received 0·002 c.c. It did not react, though susceptible.
Ox 973 received 0·02 c.c. It reacted the fourth day.
Ox 974 received 0·2 c.c. It reacted the third day.

EXPERIMENT 4. **12/12/23.** Virus Control (Ox 1050). Reacted fifth day. Bled seventh day after double inoculation with virus and serum. Morning temperature 103·4°.

Ox 1057 received 0·002 c.c. It did not react, though susceptible.
Ox 1058 received 0·02 c.c. It did not react, though susceptible.
Ox 1059 received 0·2 c.c. It reacted the seventh day.

EXPERIMENT 5. **12/12/23.** Using a liver extract of the above animal. The extract was made by mixing 100 c.c. of saline with 100 gm. of liver, and filtering through gauze.

Ox 1054 received 0·002 c.c. It did not react, though susceptible.
Ox 1055 received 0·02 c.c. It did not react, though susceptible.
Ox 1056 received 0·2 c.c. It reacted the fifth day.

EXPERIMENT 6. **20/2/24.** Using peritoneal fluid collected from Ox **1143**, which had been given **4** litres of warm saline intraperitoneally on the seventh day of reaction and 1½ hours before it was bled to death.

Ox **1191** received **0·002** c.c. It did not react, though susceptible.
Ox **1192** received **0·02** c.c. It did not react, though susceptible.
Ox **1193** received **0·2** c.c. It reacted the seventh day.

EXPERIMENT 7. **20/8/24.** Virus Control (Ox **1413**). Reacted third day. Bled fourth day. Morning temperature **105·2°**.

Ox **1421** received **0·002** c.c. It reacted the sixth day.
Ox **1422** received **0·02** c.c. It reacted the fifth day.
Ox **1423** received **0·2** c.c. It reacted the fifth day.

EXPERIMENT 8. **20/5/25.** Virus Control (Ox **1573**). Reacted fourth day. Bled seventh day after double inoculation with virus and serum. Morning temperature **104·4°**.

Ox **1580** received **0·002** c.c. It did not react, though susceptible.
Ox **1581** received **0·02** c.c. It did not react, though susceptible.
Ox **1582** received **0·2** c.c. It reacted the fifth day.

EXPERIMENT 9. **2/6/25.** Virus Control (Ox **1583**). Reacted fourth day. Bled fourth (i.e. same) day. Morning temperature **104·8°**.

Ox **1588** received **0·002** c.c. It did not react, though susceptible.
Ox **1587** received **0·02** c.c. It reacted the fifth day.
Ox **1586** received **0·2** c.c. It reacted the fifth day.

EXPERIMENT 10. **18/6/25.** Virus Control (Ox **1588**). Reacted fourth day. Bled eighth day. Morning temperature **102·8°**; recovering from mild reaction.

Ox **1593** received **0·002** c.c. It did not react, though susceptible.
Ox **1594** received **0·02** c.c. It reacted the sixth day.
Ox **1594** received **0·2** c.c. It reacted the fourth day.

Of these ten experiments the second was spoiled by one of the oxen proving to be immune, and Experiment **7** by the series of dilutions not going below the one of which **20** c.c. contained the minimal infective dose; but neither of these experiments is inconsistent with our supposition, and the remaining eight definitely support it.

We feel justified, then, in continuing this series, and if we continue to get regular and consistent results we hope to obtain enough data therefrom to enable us to construct a curve showing the rise and fall of virus concentration in the blood and, afterwards, in other tissues as the disease advances. From such data we may learn how to obtain the maximum of antigen for the purpose of hyperimmunising.

We should not like it to be thought that we were of the opinion that susceptibility is an invariable; we only think that it is constant to the extent that any complex biological phenomenon can be

constant; sufficiently invariable, i.e., not to vitiate unduly results of experiments to determine the minimal infective doses of virulent fluids. During other experiments of a similar nature to those of the above series we have met with two cases which appear to be exceptions to the rule that all susceptible oxen will react to a concentration of virus greater than the minimal infective one. In one of these cases we were dealing with blood from a beast at the height of a severe reaction, and to one ox we gave 0·0002 c.c. of this blood, and to another 0·002 c.c. The one which received the smaller dose reacted on the eighth day, while the other remained susceptible. The other case was when a susceptible ox received 0·2 c.c. of virulent blood and did not react, although its fellow reacted to 0·02 c.c. Such exceptions, provided they are infrequent, should not be considered to invalidate a biological law.

Although the hypothesis of constant susceptibility, together with the hope of getting reliable information from the minimal infective dose method of analysis, must be discarded if such exceptions as those mentioned in the preceding paragraph crop up frequently, one should not expect that if a number of susceptible oxen are inoculated each with 20 c.c. of the lowest dilution which will set up infection in that dose, that all these inoculated beasts will react; because with high dilution and consequent low concentration of virus it is unlikely that every random sample of 20 c.c. will contain an infecting unit. Thus in the following experiment, dated February 2, 1925:

> Virus Control (Ox 1520). Reacted fifth day. Bled seventh day. Morning temperature 104·8°. We considered that 0·002 would be about the minimal infective dose, so we inoculated each of three oxen with this amount. One reacted on the sixth day, one on the seventh day, while the third failed to react and remained susceptible.

In this experiment the long incubation periods indicate that the infecting doses were actually about the minimum, and so it is not surprising, and it is in no way inconsistent with our hypothesis, that the third failed to react. Even if susceptibility be not the constant we postulate, it is difficult to connect susceptibility with the course which the disease runs. It matters not whether a beast gets a minimal infective dose or a massive infecting dose, the course of the disease is likely to be the same. This is shown by our statistics which indicate that those animals which were infected by very small doses did as a rule take longer to incubate the disease, but that thereafter they died or recovered in the same proportions as do the beasts which are infected with large doses of virulent material. Since lessened susceptibility could only protect its possessor by

reducing the amount of virus which gets past the barrier furnished by this lessened susceptibility, and since the course of the disease is independent of the size of the infecting dose, therefore the onus of controlling the course of the disease is thrown on the *resistance* which the body can furnish after infection has been achieved.

Conclusions.

A distinction should be drawn between *Susceptibility*, as the opposite of Immunity, and the *Resistance* offered to the virus after it has obtained a hold in the animal body.

There is considerable evidence to show that susceptibility is fairly constant, and so without influence on the course of the disease; and that, therefore, such an expression as "low susceptibility" should be replaced by "high resistance."

The constancy of susceptibility, if proved, will allow the concentration of virus in any liquid to be ascertained by the minimal infective dose method.

THE TRYPANOSOMIASES OF MAN AND ANIMALS IN SOUTHERN RHODESIA.

By Ll. E. W. BEVAN, M.R.C.V.S.,
*Director of Veterinary Research.**

In order to introduce this subject for discussion, I venture to present a brief review of the trypanosomiases of man and animals as they occur in Southern Rhodesia.

Trypanosomes are minute unicellular animal parasites about one-thousandth part of an inch in length, which are met with in the blood and other tissues of infected animals. In the blood they may be seen swimming freely among the blood-cells, which they disturb more or less violently by their movements; it is from this characteristic that the name trypanosome (from the Greek *τρύπανον*, borer, *σωμα*, body) is derived.

These parasites are met with in all sorts of animals, birds, beasts, and fishes. In some they appear to cause no harmful effects, in others they give rise to disease. For example, there is a trypanosome of rats, particularly sewer rats, in all parts of the world, which is of very low pathonogenicity and appears to be transmitted by the rat flea; there is another in horses in certain parts of Europe, along the south littoral of the Mediterranean and elsewhere, which gives

* An introductory address delivered at a conference held at the Veterinary Laboratory, Salisbury, April 14, 1925, to consider ways and means of dealing with the "tsetse fly" and the diseases transmitted by it.

rise to a severe disease and is transmitted by coitus, hence the name "maladie du coit." There is another which causes the so-called surra of horses, camels, and other domestic animals in India, China, and the Philippine Islands, which for a long time was thought to be transmitted from sick to healthy by biting flies, but recently has been found to be cyclically transmitted by a tick of the species Ornithodorus. Indeed, more than a hundred species of trypanosomes have been discovered and christened with more or less appropriate names.

I mention this at the outset because there appears to be some idea that there is but one trypanosome—that sent for our own special benefit; and that wherever there is a trypanosome there must be a tsetse fly. Fortunately or unfortunately, both these ideas are incorrect.

In this country several varieties of trypanosome have been met with. For example I have found *Trypanosoma nanum* in the blood of a horse recently returned from a hunting expedition near the Zambesi, *T. capræ* in the blood of a goat in the Gatooma district, *T. theileri* and *T. vivax* rarely in cattle, and *T. simiæ* in pigs; but the two most commonly encountered are *T. congolense var pecorum* in cattle and *T. brucei var rhodesiense* in man, game, and domestic animals. The former is far more frequently found and is the chief cause of the loss of cattle, which we are here to lament and, if possible, to prevent. I may therefore be forgiven if I deal with this parasite a little more fully.

It may be remembered that in 1908 Montgomery and Kinghor, working in Northern Rhodesia, discovered the presence of *T. dimorphon*, and suggested that it may be transmitted from sick to healthy by biting flies other than the tsetse. This statement caused considerable alarm, and troops of cattle from the north were placed in quarantine pending further inquiry. On my return from the Pasteur Institute in 1909 I carried out investigations chiefly in the Hartley district, and as a result drafted an interim report, in which I identified the trypanosome of cattle in that area as *T. pecorum*. Recently Thomson has expressed the opinion that it belongs to the group *T. congolense*, but in order to emphasise certain peculiarities I continue to call it *T. congolense var pecorum*.

This parasite causes a subacute disease in cattle, which in some circumstances becomes very acute. It is well known to all of you that during the dry season cattle may be infected, although they remain apparently in good health so long as grazing is plentiful; when the cold weather comes and grazing is scarce they then begin to fall away in condition and many die. Cattle exhausted by overwork and under-feeding can ill resist the disease. The majority of

deaths occur at the commencement of the rains. It sometimes happens that comparatively fat cattle, when exposed to the first rains, die suddenly, their death being attributed to quarter-evil or anthrax, so rapidly does it occur. On examination these are found to have died of trypanosomiasis.

It is said that sheep and goats are to a large extent resistant, and that mules, donkeys, and horses are less susceptible than cattle. Certainly in 1909 mules and donkeys travelled with impunity throughout the Hartley and Gatooma districts, and healthy goats and sheep were met with where no cattle had survived.

The infectivity of the parasite, however, appears to vary with passage. For example, in order to study the subject at the laboratory I inoculated small animals from infected cattle without success. It was not until I had taken a certain white rabbit to Hartley on three visits and had inoculated it on each occasion that its resistance broke down and it became infected. From that rabbit it was henceforth easy to infect others. Again, the disease was difficult to establish in sheep, but when a certain animal's resistance was reduced by repeated injections of blood from rabbits, the trypanosome established in it could be transferred to other sheep. The disease also became exalted in virulence by such passage, and whereas the first sheep lived three months, later on sheep died in less than half that time. Thus we see the parasite can become accommodated to unusual hosts, and can increase in virulence by passage from host to host.

This trypanosome is said also to be present in game, but I have to admit that I have only once proved it to be present, namely, in wart-hogs. On a certain shooting expedition, remembered by some of you, these constituted our only victims, and from them I inoculated rabbits, some of which became infected with a trypanosome, which rightly or wrongly I regarded as *T. pecorum*. The part played by game is a very important question which will no doubt be keenly debated by you all later in the day.

With regard to the second important trypanosome, it is one bearing the undesirable name *T. rhodesiense*. It is a very unfortunate name, and one against which I have frequently protested, because it is not even yet known whether this trypanosome is not merely a variety of the *T. brucei*, a parasite named after Bruce who discovered it in animals in Zululand in 1896, and which is now known to be present in other parts of Africa.

The so-called *T. rhodesiense* was first seen in man by Dr. MacKenzie, and was first described in an article by me contributed in 1910 to the *Journal of Comparative Pathology and Therapeutics* under the heading: "Note on the Passage of a Human Trypanosome

through Domestic Animals." It is not a parasite commonly met with either in animals or man in this country, but by reason of the fact that deaths have been caused by it in both, it is of serious importance. It has also been met with in game. I have found it in reed-buck and water-buck. Kinghorn and Yorke (1912) found that 16 per cent. of the game examined by them in the Luangwa Valley were infected.

The most important feature of this parasite is that whereas in the past the trypanosomiasis of man—the so-called sleeping-sickness—was transmitted by *Glossina palpalis*, this trypanosome can be transmitted by the *G. morsitans*, the common tsetse fly of this country.

But in spite of all the alarming theories which have been propounded, and the reams of alarmist articles which have been written, it is comforting to reflect that it is a very very rare thing for man to become infected. How, why, when, and where this happens, others present may be able to explain.

In South Africa the trypanosomiasis of animals, "Nagana," has rightly or wrongly always been associated with the tsetse fly; this association has been forcibly impressed upon native and European alike, by the very practical observation that when their animals were exposed to the bite of this particular fly they died. The old records of the hunters, missionaries, and transport riders all emphasise this point. It remained for Bruce, in the report of which I have an original copy before me, dated Ubombo, Zululand, May 29, 1896, to describe :—

1. "The tsetse fly, with experiments designed to show the part that fly takes in the causation of the disease."
2. "A description of the Hæmatozoon or blood parasite, which is the cause of the disease."
3. "The results of experiments having for their object proof of the connection which exists between the big game and the spread of the disease."
4. "A description of the disease as it affects domestic animals, with illustrative cases."
5. "Inoculation and feeding experiments to show the communicability of the disease from affected to healthy animals."
6. "Treatment of the disease, prophylactic and curative."

We may well ask, How far have we got since then? I am afraid some of you will say, not very far.

Bruce demonstrated that the tsetse fly was "able to retain its infective power for at least twenty-four hours," but later the same observer showed that mechanical transmission can take place if the

time of the transference of the flies from the infected to the healthy animal is not longer than two hours. In 1909, however, Kleine discovered that after an interval of twenty days, or more, after taking up infected blood, the tsetse became infective and thereafter remained capable of infecting. The length of the interval and the percentage of flies becoming thus infective was found later to depend upon temperature. The number of infective flies was thought to be under 10 per cent., but recent observations indicate that the proportion is greater. As tsetse flies require to feed every few days, an infective fly can, of course, do an enormous amount of harm.

But is the transmission of trypanosomiasis entirely dependent upon the tsetse fly, and can infection be transmitted from sick to healthy by other means? This is a very important question, and one upon which you will be asked to express an opinion.

Of course, the mechanical transmission of a trypanosome in certain circumstances is extremely easy. It is so easy that it has always to be carefully guarded against when carrying out experiments in the laboratory. A drop of infected blood from one animal if transferred rapidly to the wound of another may give rise to infection. The second animal may be the operator. It is easy to conceive that a wound having been inflicted by a thorn, a blood-sucking fly, or other agent, blood might escape containing trypanosomes, and that these might be carried by flies to the wound of a healthy animal, which might thus become infected. This being the case, it is somewhat surprising that trypanosomiasis so rarely occurs outside tsetse-infected areas. I could quote numerous instances of infected and healthy cattle having run together in close contact, in the presence of innumerable biting flies, without any secondary cases occurring. I have known infected horses standing side by side with healthy horses in a stable where Stomoxyidæ were so numerous and vicious that the poor animals stamped their shoes off with constant kicking, and yet the in-contacts remained healthy: I have known infected pigs with *T. simiæ* numerous in their blood, constantly bitten by Stomoxyidæ and pig-lice, in styes separated only by wire from other pigs, none of which became infected. On one occasion my small animal rooms were invaded by fleas, but infection remained limited to those animals inoculated. My own impression, based upon nearly twenty years' experience in this country, is that, so far as Southern Rhodesia is concerned, mechanical transmission rarely if ever happens because the conditions which render it possible seldom occur. These conditions are, a plentiful supply of tyrpanosomes in the peripheral blood of the infected animal (*T. pecorum* is never present in great numbers), close contact between sick and healthy animals, special climatic

conditions, such as warmth and moisture, which enable the parasite to live in the blood, and very rapid transmission so that the blood may not have coagulated in transit from one animal to another.

But the fact remains that there is a risk of mechanical transmission, and one which must be taken carefully into consideration. In Zanzibar, in the absence of tsetse, trypanosomiasis occurs, and I believe I am right in saying that the trypanosome is *T. congolense*. It may, however, be one which has become accommodated to such a method of transmission.

The problem before us therefore is to determine (1) whether the local trypanosomiases originate from the game which act as " carriers " or " reservoirs " of infection ; (2) whether the parasites are transmitted from infected to healthy by the tsetse only ; or whether (3) infection can be transmitted mechanically ; and if so (4) whether such transmission so commonly takes place as to constitute a real and practical danger. Prophylactic measures must of necessity be based upon these considerations.

Based, I presume, on the well-known association between the " fly " and the big game, certain organised attacks have been made upon the latter, with what results we shall no doubt learn. There seems, however, some doubt as to whether tsetse are entirely dependent upon the blood of wild animals, and whether in their absence they will not seek and obtain suitable nourishment from man and his domestic stock. Also it has been suggested that the disturbing of the game from their natural haunts may result in the dispersal of the various strains of trypanosomes which they harbour. For example, *T. brucei var rhodesiense*, which as far as we know is at present limited in its distribution, may be conveyed into neighbouring tsetse-infested country where previously only the *T. pecorum* was known to exist.

The question as to the association between tsetse and game is probably the one which will interest you most, as I see before me those who hold entirely opposite views on the subject.

I think it will be conceded that in most outbreaks infection originates from the tsetse, therefore steps should be taken to eliminate the tsetse. But how is this to be done ? It would at first sight appear as hopeless a proposition as the arresting of the progress of the waves appeared to Canute. That, however, should not deter us if it be decided that civilisation or the tsetse must go.

It may be pointed out that the tsetse is a slow breeder ; it does not lay eggs by the thousand as do the ticks, or other Diptera, but produces at birth " a single full-grown larva, which, while retained within the oviduct of the mother, is nourished by the secretions of

special glands, and on being born crawls away into a hiding-place and immediately changes into a pupa." Once born, the larva has to seek shelter. To Jack belongs the credit for having first drawn attention to the manner in which certain positions are selected by *Glossina morsitans*, the local variety of tsetse fly. Chorley has shown that when pupæ "go to earth" as it were, elsewhere, many are actually destroyed by the heat of the sun. Cameron found that they were devoured by certain birds. Lamborne and Chorley have demonstrated that an enormous proportion are parasited by other insects such as the Syntomosphyrum and certain Bombalid flies.

For these and other reasons, which will be dealt with more adequately by the distinguished entomologists here to-day, it occurs to me that the life of the tsetse, like that of the proverbial policeman, "is not a happy one." Moreover, the positions suitable for the perpetuation of the tsetse are not ubiquitous—indeed, they are very localised, and I believe investigation many prove that they are even more circumscribed than they appear at first sight. Therefore, to me, the eradication of the tsetse appears a less formidable proposition than was the eradication of the tick before the era of dipping.

It is generally admitted that the tsetse disappeared, or markedly decreased, after the rinderpest. Some explain this on the grounds that their natural food was entirely taken from them. This, however, was not always the case, for many varieties of wild animals, including antelopes, wildebeest and zebra, were not affected and presented a possible source of nourishment. The recent review by Fuller on the "Tsetse in the Transvaal and Surrounding Territories" is particularly interesting and deals very fully with this question.

The idea that the blood of animals sick or dead from rinderpest carried a poison harmful to the tsetse is disproved by the experiments which Montgomery informed me he carried out at Kabete, where he fed tsetse on animals infected with that disease without ill-effects to the fly. I am inclined to the opinion that the explanation may lie in the fact that the tsetse after the rinderpest was compelled to imbibe blood from other than its usual or predilection hosts, and that this did not prove suitable for the nourishment and subsequent development of the larvæ. Mr. Chorley kindly provided me with several hundred pupæ, of which more than 70 per cent. successfully hatched out in my laboratory. The adult flies immediately and contentedly accommodated themselves in certain boxes which I had devised for them, and fed freely on rabbits. A large number of the larvæ were born from them and proceeded to the pupal stage, but none reached the adult stage. It would appear that while rabbits'

blood is sufficient for the nourishment of the adult fly, it is lacking in certain elements essential for the propagation of the fly.

In addition to the destruction of game, deforestation has been suggested as a means of reducing the number of tsetse. This is based upon the well-known theory that tsetse require shade. The removal of all shade-supplying trees from tsetse-infested country would appear a colossal undertaking; but with settlement and the spread of civilisation this may take place gradually and almost automatically—small comfort, it is true, to the pioneer who bears the heat and burden of the day. One incident, however, gives me reason to believe that this method may be less difficult and more successful than some of the others suggested. In 1909 Captain Thornton and I surveyed the Hartley and Gatooma districts, and came to the conclusion that while at certain times of the year tsetse made their way along the head-waters of the Surasura River and attacked cattle even as far away as the Eiffel Blue Mine on the southern side of the railway, during the dry season they were localised to certain pools in the vicinity of Fopogena's Kraal. This state of affairs continued until 1912, when certain transport riders, in carrying out wood contracts with the Cam and Motor Mine, cut down the timber in this neighbourhood, and, I believe I am right in saying, the district has been free from tsetse since. This, I know, is attributed to the shooting and driving away of the game, but the transport oxen and the many herds in the district should still have provided an ample supply of food.

These problems, however, are for the entomologist; my immediate business appears to be the keeping of your animals alive until the entomologist has solved his difficulties. This brings me to the subject of treatment.

In 1908, when studying at the Pasteur Institute, Paris, it was my good fortune to meet Major Kerandel, an officer of the French Army who had contracted sleeping-sickness. He had been treated by Sir Patrick Manson in London with alternate doses of tartar emetic and atoxyl, but the former was introduced sub-cutem and gave rise to serious pain and sloughs. Notwithstanding this the patient improved rapidly and returned to Paris. Later, however, he suffered from a relapse and was treated by a French physician, who injected the antimony into the vein. Kerandel, who was cured by this treatment, gave me the full particulars, and suggested that I should apply it to trypanosome-infected cattle. On my return to Rhodesia I carried out many field tests of this treatment,* and was

* As far as I am aware this was the first time this treatment was applied in South Africa to animals suffering from trypanosomiasis.—Ll. E. W. Bevan.

astonished to find that cattle would tolerate enormous doses of the emetic, but that it had to be injected very carefully into the vein, for if any of it made its way under the skin it gave rise to severe swellings, abscesses, and sloughs.

Having no atoxyl, I used methyl arsenate of soda as the alternate drug, and later a preparation sent me by the late Professor Ehrlich, the nature of which was not divulged. It was not for a long time, however, that I came to the conclusion that it was the antimony rather than the arsenic which exerted the specific effects upon the trypanosome, the arsenic being useful in counteracting the anæmia and loss of tone associated with the disease.

But in practice, the administration of antimony potassium tartrate is by no means easy. The animal has to be caught and thrown, the jugular vein has to be raised, and the solution has to be slowly injected into it, care being taken that not a drop escapes into the subcutaneous tissues. Care has also to be taken that the water in which the drug is dissolved is chemically pure; in one instance ten animals were killed through the neglect of this precaution. It was considered wise to repeat the injection several times at an interval of five days or a week. Where many cattle were involved the work of treating them in this way was a serious matter. To obviate this I have recently prescribed a combination of antimony and arsenic which can be injected under the skin without damage to the subcutaneous tissues and the cattle can be treated without being thrown. There is no reason why the effects of this treatment should not be as good as with the old method.

It is true that swellings and abscesses do sometimes occur, but these, I think, are generally due to the introduction of foreign bodies and pus-forming organisms during the operation. During the treatment the animal has to be generously nourished in order to build up its depleted tissues. I have found it necessary to print on the label of the bottle containing the injections: "This medicine must not be regarded as a substitute for food and water." I think from recent correspondence that this little hint has had the desired effect.

But this treatment, although useful in keeping a high proportion of infected animals alive in tsetse areas, is by no means perfect. Although it may cure some, that is, sterilise them of the parasite, the majority are not actually cured, but only rendered "tolerant" to the parasite, which they continue to harbour in the blood although unharmed by it. How this tolerance is brought about is a very interesting question, but one which can scarcely be considered here. The whole question of immunity is a very complicated study.

It is a well-known saying that "prevention is better than cure,"

and, freely admitting the defects of the treatment, I am endeavouring to discover a means whereby animals may be protected against infection. Much has been said and written about Bayer 205, which was heralded as the panacea for all the evils due to trypanosomes. Unfortunately these optimistic forecasts have not been fulfilled. Experiments in the Belgian Congo and elsewhere have shown that this drug is less successful in the treatment of cattle than of man. But it exerts certain very remarkable effects quite distinct from those produced by the antimony salts, and these lead one to hope that by a careful study of the manner in which it acts a clue may be obtained leading to a method of protecting rather than of curing our animals.

I have often thought that systematic dipping might be useful in helping to protect cattle against infection, and possibly in reducing the number of tsetse. It occurred to me that the residual arsenic which is said to be present in the lower layers of the skin of regularly dipped animals might act in several ways: (1) in killing the trypanosome injected into the arsenic-containing layer; (2) in poisoning the fly which imbibed the arsenic when feeding; (3) in sterilising the fly containing trypanosomes in its proboscis, gut, or elsewhere; (4) in rendering the blood unsuitable for the nourishment of the larva within the fly. All these ideas were based upon the effects of dipping upon the tick.

However, I was told that the proboscis of the fly was so long that it penetrated below the arsenic-containing layer. It occurred to me that the fly did not require a beefsteak at a meal, but merely a drop or two of blood. I therefore watched them feeding, and found that although they would sometimes introduce their proboscis deeply into the skin and underlying tissues, they would later carefully withdraw it until they arrived at a certain depth from which blood could be obtained. It was not for that reason that I was disappointed in my few experiments in feeding flies upon dipped cattle, but for other reasons which I need not discuss here. I have reason to believe that by an alteration in the composition of the ordinary cattle-dip better results may be obtained.

A series of experiments recently carried out at this laboratory have proved very conclusively that regular dipping has a very marked effect upon the course of the disease in the infected animal, and these observations must be continued on a larger scale.

But I would draw attention to the extreme difficulty of carrying out research into a dozen subjects at one time. Research requires undivided attention and concentration on the subject under investigation. The official research officer is an unfortunate being: he is

expected to turn out discoveries like sausages from a machine. He is also handicapped by the official routine of his office.

In my opinion the investigation of the problem of trypanosomiasis, or rather the many problems which this subject presents, should be the whole-time work of a team of specialists free from the cares and worries of official and other mundane matters. And this team would of necessity require the help, and profit by the experience, of the practical man.

SOME NOTES ON SURRA IN THE CAMEL, ITS TREATMENT AND PREVENTION.

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(*Continued from* p. 499, 1925.)

The Value of Sodium Antimony Tartrate in Clearing the Peripheral Circulation of Trypanosomes.

A series of experiments were carried out in an endeavour to find out how long it takes to clear the peripheral circulation of trypanosomes, by administering varying quantities of 1 per cent. solution of sodium antimony tartrate at short intervals, and examining the blood shortly after each intravenous injection, as follows :—

Experiment A.—Camel 346 + about five trypanosomes per field at 11 a.m., February 20, 1924, temperature 103°. 12.20 *p.m.* : Intravenous injection of 20 c.c. of a 1 per cent. solution of sodium antimony tartrate. 12.25 *p.m.* : Blood examination (live preparation), trypanosomes about the same number (+ five per field), very vigorous. 12.30 *p.m.* : Intravenous injection of 30 c.c. of a 1 per cent. solution of sodium antimony tartrate. 12.40 *p.m.* : Blood examination, no trypanosomes found (blood rendered apparently negative to the microscope within twenty minutes). 1 *p.m.* : Blood examination revealed no trypanosomes. 1.15 *p.m.* : Intravenous injection of 50 c.c. of a 1 per cent. solution of sodium antimony tartrate. 6 *p.m.* : Blood examination was negative to trypanosomes.

Notes on Experiment A.—The peripheral circulation was apparently cleared of trypanosomes with 50 c.c. of sodium antimony tartrate in a 1 per cent. solution within twenty minutes. This result, however, was only apparent, for no injections were given after those quoted above, and trypanosomes were found in the blood eleven days afterwards and remained in evidence for five days ; showing that 100 c.c. of a 1 per cent. solution given within one hour does not destroy all trypanosomes, but that some persist, and having multiplied are in evidence in the blood some few days later.

This camel suffered no inconvenience during the experiment, feeding well on the evening of the 20th of February, 1924, and its temperature was normal on the morning of the 22nd of February, 1924. Regular treatment with sodium antimony tartrate was continued on the 7th of March, 1924, and he was eventually cured, having received 32½ grammes of sodium antimony tartrate.

EXPERIMENT B.—To determine the length of time taken, and the quantity of 1 per cent. solution of sodium antimony tartrate required to render a surra-positive camel, with its blood teeming with trypanosomes, apparently negative. Further, to find out how long the blood of such a camel rendered negative will remain apparently free of trypanosomes in its peripheral circulation :—

Camel No. 198, February 22, 1924. 11.30 *a.m.* : + surra (over twenty per field). 11.50 *a.m.* : Intravenous injection of 20 c.c. of 1 per cent. sodium antimony tartrate. 11.55 *a.m.* : Blood examination + swarming and vigorous. 12 *noon* : Intravenous injection of 30 c.c. of 1 per cent. sodium antimony tartrate. 12.5 *p.m.* : Blood examination + ten per field, rather sluggish in movement. 12.10 *p.m.* : Intravenous injection of 20 c.c. of 1 per cent. sodium antimony tartrate. 12.15 *p.m.* : Blood examination + five per field, movements of trypanosomes slow. 12.20 *p.m.* : Intravenous injection of 30 c.c. of 1 per cent. sodium antimony tartrate. 12.25 *p.m.* : Blood examination + one trypanosome only found in the live blood preparation, movement slow. 12.30 *p.m.* : Intravenous injection of 50 c.c. of 1 per cent. sodium antimony tartrate. 12.35 *p.m.* : Blood examination + one trypanosome only found ; movement very slow. 12.40 *p.m.* : Intravenous injection of 50 c.c. of 1 per cent. sodium antimony tartrate. 12.50 *p.m.* : Blood examination — no trypanosomes were found.

Notes on Experiment B.—It will be seen that the blood of a camel containing more than twenty trypanosomes per field (× one-sixth live preparation) was rendered apparently negative with a total quantity of 200 c.c. of a 1 per cent. solution of sodium antimony tartrate (i.e. 2 grammes), given in small doses at ten-minute intervals, within one hour, the camel suffering no inconvenience in consequence.

No further intravenous injections were given, and the camel was kept under daily blood-test. The blood remained negative for eleven days, but there was a return of trypanosomes on the twelfth day of observation.

It would appear from Experiments A and B that 1 or 2 grammes of sodium antimony tartrate does not destroy all trypanosomes in a case presenting numerous trypanosomes, and that certain of them persist, and injections must be continued till all trypanosomes have

been destroyed, otherwise a relapse will occur. In this connection the case of Camel No. 126 is of interest. After receiving 16 grammes of sodium antimony tartrate, treatment had to be suspended for one month on account of sickness, and during that period, as this camel did not present trypanosomes in its blood, treatment was continued Other experiments on the above quoted lines were carried out which gave much the same results.

Camel No. 235 (+ twenty per field) was given 4 grammes of sodium antimony tartrate 1 per cent. solution within three days. The animal was rendered apparently negative within one hour after receiving ½ gramme, but relapsed in fifteen days after the last injection. He fainted after receiving the first ½ gramme, and remained unconscious for fourteen minutes. After recovery, he suffered no further inconvenience whilst undergoing the experiment till the return of trypanosomes to the peripheral circulation.

The case of camel No. 710 is also of interest, in that he presented trypanosomes in his blood to the number of about fifteen per field. He was given an intravenous injection of a 100 c.c. 1 per cent. solution of sodium antimony tartrate and his blood, when microscopically examined fifteen minutes later, appeared negative to trypanosomes. This camel showed no signs of fainting or giddiness during or after the injection, and was eventually cured, having received 30 grammes of sodium antimony tartrate.

It appears that although fainting may occur with intravenous injections of sodium antimony tartrate in camels presenting large numbers of trypanosomes in their blood, it is much less likely to occur than when potassium antimony tartrate is used, and the sodium salt may be said to be very much less dangerous from this point of view.

Surra-positive camels that do not present trypanosomes in their blood when examined microscopically may be given an initial dose of 200 c.c. of a 1 per cent. solution of sodium antimony tartrate with safety.

The Dosage Employed in Treatment by Sodium Antimony Tartrate.

It has been previously recorded that no standard dosage may be laid down in the case of treatment by potassium antimony tartrate, and that in estimating the doses for intravenous injection one has to be guided by close observation of the individual case. In the same way it is not possible to lay down a definite scale of doses for all cases where sodium antimony tartrate is the curative agent employed.

The degree of toleration to the drug is not the same in all camels. This may be due to some extent to the degree of damage done to the system by the disease ; on the other hand it may bear relation to idiosyncracy. However, it may be asserted that if the blood of a given camel is microscopically negative to trypanosomes at the time it is required to give the initial injection, a dose as large as 200 c.c. of a 1 per cent. solution of sodium antimony tartrate (i.e. 2 grammes) may be given with safety, and provided the camel is strong and in fairly good condition, this dose is usually well tolerated every third day until a total of 30 grammes or more of sodium antimony tartrate has been reached.

Thus with the average surra-infected camel the course of treatment

Camels after a Course of Treatment with Sodium Antimony Tartrate. The three camels in the background came into Musth during treatment, but the good muscular development of their hind limbs is well demonstrated.

is reduced from seven weeks to two months or more in the case of treatment by tartar emetic to about four or five weeks' duration in the case of the sodium salt. Moreover, with the administration of larger quantities of the drug, the disease is more rapidly brought under control, and the effects of toxæmia is soon very much reduced. This is exampled in the rapid improvement in general health and condition observed soon after treatment is begun, i.e. when the 200 c.c. 1 per cent. solution standard dose is kept up.

However, at any time should adverse symptoms be displayed, in a given case during such a course of treatment as described above, the animal must at once be rested from treatment and magnesium sulphate administered in large doses, and not until it has regained an apparently normal state of health should treatment be continued. It is then advisable to recommence treatment with a smaller dose, viz. 50 to 100 c.c. 1 per cent., and gradually raise the dose to the 200 c.c. 1 per cent. standard.

Caution should be exercised in restarting treatment, keeping in view the fact that eventually the camel will tolerate the drug with comparative comfort, whereas if treatment be continued while he is still suffering from the effects of the drug, death will possibly supervene. In illustration of the foregoing remarks, I have prepared a tabulated list, Table III, giving some account of thirty-four camels treated by intravenous injections of sodium antimony tartrate between February and June 1924, this being the total number of surra-positive camels treated at Lahore with this salt of antimony up to the time of recording these notes.

Notes on Table Opposite.—Number of camels treated with sodium antimony tartrate was 34. Number cured, 32, or 94 per cent. Number died, 2, or 6 per cent. Average number of grammes administered, 34, a maximum being 43½ and the minimum 28. The average number of days under treatment was 47·5, the longest period being 102 days and the shortest 27 days. The number of camels that lost weight, 2, and the average loss was 20 lb. The number of camels that increased in weight, 30. The average increase of these, 128 lb., the maximum increase being 340 lb. and the minimum was 20 lb.

It will be seen that as much as 40 to 43½ grammes of sodium antimony tartrate were administered to certain camels. In those cases the number of days under treatment was considerably higher than those receiving 30 grammes or thereabouts. Thirty grammes is possibly sufficient to produce a cure, unless there has been a long period of sickness during treatment. In other cases, where the number of days under treatment ran into fifty or sixty days, it indicated a period or periods of sickness during treatment.

The dosage employed was as far as possible the 200 c.c. 1 per cent. solution standard, but this was dropped to 100 c.c. or 150 c.c. 1 per cent. solution, when adverse symptoms were displayed, and there was, of necessity, rest from treatment.

When treatment was started during a paroxysm of fever and trypanosomes were found in the blood, doses of 20 up to 50 c.c. of ½ per cent. solution were given as an initial injection, and if negative to trypanosomes on the following day, 200 c.c. of 1 per cent. solution was administered and continued as far as possible every third day till treatment was completed.

The Changes that take place in Surra-Positive Blood after an Intravenous Injection of a Solution of Either Sodium Antimony Tartrate or Tartar Emetic.

From the microscopical examination of a series of stained blood-smears, taken at definite intervals of time after an intravenous

TABLE III.

Camel No.	Number of Grammes of Sod. Ant. Tart. in 1 % Solution administered during Treatment.	Number of Days under Treatment.	Result.	Increase of Body-Weight at Termination of Treatment.	Remarks.
	grammes.			lb.	
905	36½	49	Cured	280	
198	28	58	Died	—	After 28th gramme became sick and died eleven days afterwards.
1015	30	56	Cured	60	
897D	28	33	Died	—	
266	30½	27	Cured	160	Came into musth during treatment—250 c.c. of a 1 % sol. sod. ant. tart. given five times.
346	32½	45	Cured	80	*Vide* Experiment "A," p. 546.
710	30	45	Cured	20	
322	28	53	Cured	160	
404	31	52	Cured	60	
750	30	52	Cured	lost 20	
327	31½	53	Cured	40	
151	28	49	Cured	20	
703	40	51	Cured	120	
30D	31½	59	Cured	200	See Fig. 3.
792	30	40	Cured	220	
1097	31½	33	Cured	220	
437	35	47	Cured	200	
951	40	36	Cured	120	
432	35	47	Cured	40	
690	34	50	Cured	180	
803	36	42	Cured	120	
175	36	48	Cured	100	
965	34	34	Cured	80	
968	34	34	Cured	120	
5E	41	54	Cured	80	
172	37½	64	Cured	40	Œdema of chest wall and abdomen for twenty days during which time no treatment was given.
979	33½	33	Cured	100	
518	40	51	Cured	80	
208	36	49	Cured	220	
331	36½	41	Cured	340	
132	43½	102	Cured	lost 20	Sick one month from dermatitis.
695	36½	43	Cured	300	
645	30	41	Cured	20	
278	34½	42	Cured	60	
34 camels	34 grms. average	47·5 days	Cured 32 Died 2	Average 128 lb.	

injection of either sodium or potassium antimony tartrate, it would appear that trypanosomes are destroyed by actual contact with the solution in the blood stream. The disintegration caused thereby is gradual with the majority of trypanosomes, but nevertheless complete. Certain trypanosomes, however, persist, and require to be tired out with repeated administrations of the drug before they are finally destroyed.

The earliest indications of the process of destruction and disintegration may be observed most easily by examination of a live blood preparation taken fifteen or twenty minutes after an intravenous injection. It will then be found that the trypanosomes appear to become sluggish in movement and reduced in number per field, and if the blood be examined at five-minute intervals afterwards, the movement of trypanosomes will be seen to become slower and slower, till detection of individuals becomes difficult or impossible.

Series of stained blood-smears (Giemsa or Leishman), taken at ten-minute intervals after intravenous injection, are most interesting. The first indication of disintegration appears to be a transparent appearance of the cytoplasm of individual trypanosomes, i.e. the cell plasma takes on the stain very faintly. The nucleus still takes the basic stain well, but the ectoplasmic border of the trypanosomes seems to have disappeared. The flagellum, undulating membrane, and nucleolus or blepharoplast, in the majority of cases have completely disappeared, giving the trypanosomes an elongated elliptical appearance. The nucleus appears to be the last portion of the trypanosomes to disintegrate. In cases where trypanosomes have been numerous before intravenous injection, much debris of dead trypanosomes is seen to be floating about between the red cells. If stained blood-smears be examined the following day it will be found that there is an increase in the number of large mononuclear leucocytes and lymphocytes. Phagocytosis is then seen to be most active, especially in the case of the large mononuclear leucocytes, and also to some extent with the lymphocytes. These cells put out ampullæ and take up and ingest the debris, which may be seen within the cell plasma in the form of small, dark, spherical bodies. Within forty eight hours the greater portion of this debris from dead trypanosomes appears to have been cleared up.

The Changes that take place in the Blood during a Paroxysm of Fever.

The multiplication of trypanosomes in the blood during a paroxysm of fever takes place by the process known as binary fission, or simply described as a "multiplication by long division," the individual

trypanosome dividing on its long axis into two, three, and sometimes more individuals.

There appears to be two forms of trypanosome in the camel type of *T. Evansi*. (1) A long form, in which the nucleus is small, takes on the stain deeply, and with the posterior end tapering to a sharp point. (2) A shorter broad form, in which the nucleus is larger. The nucleus in this case does not take on the stain so evenly or deeply, and the posterior end is usually blunt.

The latter type appear to be principally involved as divisional forms, but this is not invariably the case. The long forms are seen in greatest number when binary fission has been or is going on very rapidly, so that the long type may be said to be the younger of the two forms, and perhaps they, in their turn, take on the characteristics of the short, broad form prior to binary fission.

The process of binary fission is gradual. One notices first of all an elongation of the nucleus, which may later divide into two, three, or more nuclei. The nucleolus duplicates or triplicates, as does the undulating membrane according to the number of trypanosomes the individual is to give rise to.

As trypanosomes increase in number, there appears to be an increase in the number of large mononuclear leucocytes and lymphocytes, and phagocytosis becomes more active. They may be seen, more especially the large mononuclear leucocytes, taking up and ingesting certain trypanosomes and also debris of any trypanosomes that have degenerated and disintegrated.

The phenomenon of the rapid appearance and disappearance of trypanosomes in the blood still appears to carry a certain amount of mystery with it. From time to time writers have described involution forms, latent or rest forms in surra trypanosomiasis, and it seems highly possible that a cycle takes place, within the body or blood stream, as in the case of *T. Cruzi* of humans, and that the trypanosome is but a phase of the protozoan in the body of the host. Further investigation is very much needed in this direction, for the importance of being able to diagnose surra between the paroxysms of fever, which often occur at such long intervals when the trypanosome is usually not to be demonstrated, cannot be over estimated in dealing with this disease.

(*Concluded.*)

Clinical Articles

DOUBLE CERVIX IN A MARE.

By W. E. ARMSTRONG, B.V.Sc.,
Wigton, Cumberland.

CASES of double cervix in mares do not appear to be common, as this is the first case I have encountered. I only regret that circumstances did not allow of further examination of the internal genitals, but I hope to do so at some future date.

Whilst examining a mare for sterility, I was asked to examine a second mare as she had something in her passage. This "something" was a thin piece of membrane stretching from the roof to the floor of the vagina. Inserting the hand further into the vagina, the double cervix could be felt, the thumb being inserted into one cervix and the second finger into the other cervix. The thumb and finger met anteriorly, showing that the cervices opened into one uterus.

FRACTURE OF THE HEAD OF THE EXTERNAL SMALL METATARSAL BONE IN A PONY.

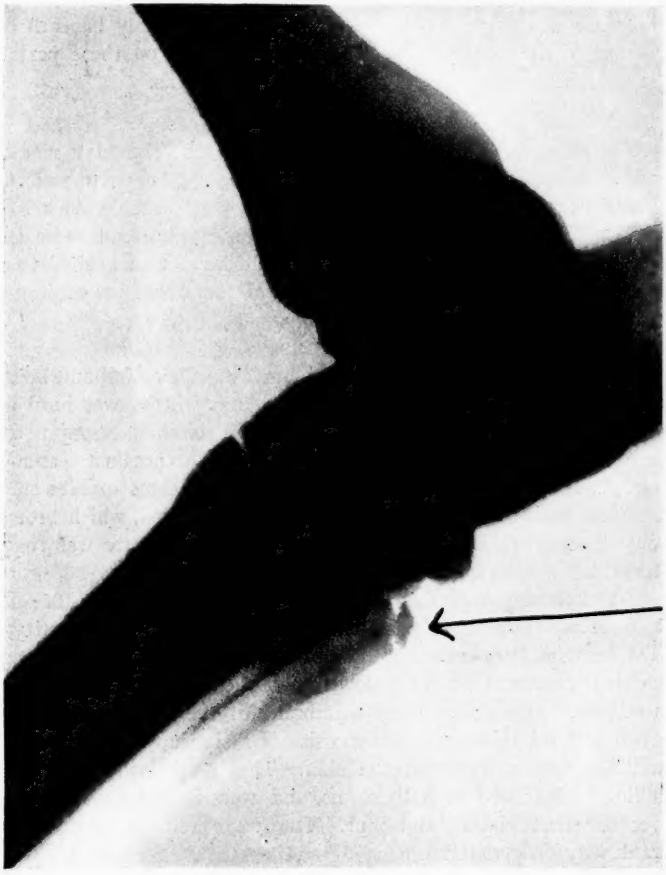
By J. F. D. TUTT, M.R.C.V.S.,
Winchester.

THIS case is recorded chiefly in view of the fact that X-ray enabled a satisfactory diagnosis to be made, and showed an extremely interesting skiagraph.

The subject was a pony, belonging to a local radiologist, and was turned out in a meadow, and it was concluded that the pony fell into a ditch, and that the fracture occurred during its struggles to get out.

When seen an hour afterwards, it was found that the near hip was bruised and swollen, and the pony was unable to put the limb to the ground. It could lie down and get up with an effort. Tenderness was noticeable on pressure round the hock, but no swelling appeared here until about three days later. No crepitus could be noted, and hot fomentations and later antiphlogistine was applied.

The owner suggested X-raying the hock joint with a portable X-ray apparatus that he had, and with the interesting result shown by the accompanying skiagraph, the head of the small external metatarsal bone being distinctly fractured and detached.



FRACTURE OF A METATARSAL BONE OF A PONY.

Treatment later consisted in two applications of biniodide of mercury blister at intervals of three weeks, and at the time of writing the pony is going on well.

Fortunately, slings were not necessary.

A CASE OF JAUNDICE IN THE HORSE.

By M. CRAWFORD, M.R.C.V.S.,
Ceylon.

Subject.

Imported four-year-old thoroughbred mare in full training. The mare was a partial "dry-sweater," sweating only beneath and in front of the saddle. She did not sweat at all on those parts of the body behind the saddle, save between the thighs.

History.

The mare was taken to the racecourse on a Thursday morning and given a fast trial gallop of 6 furlongs. She went well and pleased her trainer. On being taken back to the stable she refused food and water. The respirations were hurried, but not more than usual after a gallop. She refused food all day. I was called to see her about eight o'clock in the evening and found her presenting the following symptoms.

Symptoms.

Temperature 105·6° F. Respirations fast. Pulse full and bounding. The dung, of which she had passed very little, was hard and coated with mucus. Urine scanty and high coloured. Conjunctival membranes congested. The mare was given 2 ounces spts. ammon. aromat., 2 ounces spts. æther nitrosi, and 2 drachms quinine sulph. A copious warm water and soap enema administered, which brought away a considerable quantity of hard fæces. The mare was rugged up and left for the night.

The following morning (Friday) the temperature was 105·4° F. Respirations only slightly faster than usual. A small quantity of hard fæces and some dark yellow urine had been passed. The conjunctival membranes were yellow in colour, as were also the vaginal membranes. On the mucous membrane of the nasal septum were two bright yellow spots about the size of a sixpence. Urticarial-like swellings were noticed on the skin. They varied in size from a shilling to that of two half-crowns, and were irregularly distributed over the neck, back, and hips. The mare refused all food and drank very little water.

Although piroplasmosis has never been recorded in horses in Ceylon, this disease was suspected and the blood examined, but no parasites were found. The condition was diagnosed as acute biliary congestion. The mare was given 2 drachms calomel, 2 drachms quinine sulph., and Glauber's salts added to the drinking water. A copious warm saline enema was given in the morning and repeated in the evening.

On the third day of the illness (Saturday) the temperature was 104·6° F., and the yellow coloration of the mucous membranes and urine was intense. The urine contained much mucus. The mare ate a little tender grass and drank some water. The urticarial-like swellings were still present in the same positions. Two drachms calomel were given and saline enemata repeated.

On the fourth day (Sunday) the mare looked somewhat brighter and took a very little tender grass, some carrots and apples, but refused bran mashes. The temperature was 105·4° F. The fæces were softer. The urine contained much mucus and was brilliant yellow, as were the membranes. The urticarial-like swellings were still present. The skin seemed to be very tender, as over the zygomatic ridges where the head collar touched it the hair was removed along with the superficial layers of the skin, leaving slight excoriations in which the yellow colour was noticeable. Similar excoriations were found on the hind legs where bandages were applied. Two ounces sodium salicylate were given, and as the mare was very difficult to drench this was given as an electuary, and unfortunately produced irritation and swelling of the lips and gums. The mare was drinking more water now, and to all the water given Glauber's salts were added (2 ounces to the bucket). Two drachms potassium nitrate were also given.

On the fifth day (Monday) the temperature was 103° F., and the mare was much brighter and seemed inclined to take some food, but on account of the soreness of the gums did not eat much. She was given 1 measure bran, and ¼ measure linseed as a sloppy mash, and took it slowly. Glauber's salts in the drinking water was the only medicine given.

On the sixth day (Tuesday) the temperature was 102° F. The yellow coloration of the membranes was much less and the mare had regained her appetite. She was confined to light bran and linseed mashes with a little tender grass. The urticarial-like swellings were still present but much less prominent. The urine was still deep yellow in colour and contained much mucus.

On the seventh day (Wednesday) the temperature was 101° F. and the mare practically normal. The mucous membranes had quite lost the yellow colour, but the urine was still yellow. The urticarial-like swellings had disappeared, and the appetite was regained.

During her illness the mare had lost very little condition.

Cause of the Illness.

In conversation with the trainer I learned that the mare was being prepared for a race, and was a very greedy feeder. She had

been given more oats than any other horse in the stable. I was also informed that this mare required very little work to get her into racing condition. It would appear that the heavy feeding, with probably insufficient work, had overtaxed the liver, and the fast gallop had caused congestion of this organ with an increased production of bile.

A feature of the case was the rapidity with which the intense yellow discoloration of the membranes and urine was produced, and the equal rapidity with which it disappeared. It may be noted that the nasal septum was not diffusely coloured, the yellow coloration being confined to the two spots mentioned above. As no evidence of abdominal pain was shown at any period of the illness, it is unlikely that a gall-stone was the cause of the condition.

DIGITAL IRRITATION OF OS UTERI IN THE COW.

By O. STINSON, M.R.C.V.S.,
Appleby.

Veterinary surgeons are frequently called upon to treat cases a week or more after calving, which have an accumulation of putrid material in the uterus, the cow straining usually, but with no beneficial result. The cases referred to are those where the external os uteri is almost completely closed, and in which forcible opening of the os is contra-indicated. In most of these cases if the os were more or less dilated the condition of affairs would be the same, as the straining forces the partially contracted uterus up against the collapsed walls of the vagina, so that the accumulation cannot get through the os. The writer has found that excellent results can be got in these cases by manipulation of the os and vagina as follows. If the os is completely closed, the two first fingers are got through so that they can be opened like a pair of forceps, the internal os is irritated considerably by hooking them into the womb, as it were, the cervix being forcibly brought into the vagina several times in quick succession ; immediately the cow strains the fingers are opened forcep fashion, and in many cases a piece of placenta is felt up against the fingers ; the fingers are brought together, and the membrane is brought away by traction. After this, or instead if there is no membrane to come away, the process is repeated ; but just as the patient strains the opened fingers are brought into the cervix to keep it open while the fluid contents escape under the pressure of the strain ; this can be repeated in many cases until all the accumulated material has come away. There can be no doubt that there are a lot of cases of

this kind where forcible dilatation of the os is undesirable ; at least one condition likely to follow is sterility. When dilatation is indicated for serious womb treatment, the best results have been always achieved, but when the treatment can be carried out satisfactorily without it, it is best left undone. When the accumulation has been got rid of the practitioner's favourite antiseptic can be inserted, and, in spite of its disadvantage, pulv. iodoform (in capsules of size according to the size of opening in the os) will want a lot of beating, from half to one ounce is a useful quantity. The writer has never known of a case of poisoning from iodoform, and an ounce a day has been used for three or four days in some particular cases. Whatever is thought of the above method of treatment, it can do no harm ; the os can be forcibly dilated afterwards should the practitioner think he can do better. Frequently when the os and cervix are well dilated the cow is incapable of ridding the uterus of its contents ; as explained above, the os is forced up against the wall of the vagina. In these cases the hand is introduced into the uterus, the fist closed, and the os brought into the vagina with the closed hand like a ball fitting into the internal os ; as straining is brought about the hand is brought out quickly with the fingers spread out and the hand opened, the fingers being kept in the external os while the hand holds the vagina from collapsing, so enabling it to hold whatever may be expelled from the womb. This can be repeated as necessary until all the fluid, etc., has been got rid of. Before commencing such manipulations it is advisable to thoroughly disinfect and well lubricate with soap the vagina and os besides the vulva, as vaginitis is not an unknown condition following prolonged manipulation through the vagina. The writer has certainly come across cases in which it is impossible to excite straining by irritation of the os ; in these cases lubrication, syphoning, and scooping out with the hand generally answer well. Practitioners who do not irrigate after removing the placenta in the cow would be well advised to irritate the os after the membrane has been removed, and to have the hand ready to act as a scoop to assist in getting rid of as much fluid as possible, and in this way getting immediate contraction of the uterus after the membrane has been taken away. In the writer's opinion this is important.

MERCURIAL POISONING IN A DOG.

By J. F. D. TUTT, M.R.C.V.S.,
Winchester.

SUBJECT.—Retriever dog, four years old.

HISTORY.—The owner phoned me up one evening, stating that

his dog had been ill for about a week. He stated that its gums had been almost black, profuse dribbling from the mouth, and that it had been listless and off its food. He had given it some of Rackham's condition pills, and the blackness of the gums had nearly gone, except just at the edges of the gums, but that its feet were very swollen and tender, also its mouth, and would I send something to put it right. This I naturally declined to do without seeing the dog, and I accordingly saw it the following day.

SYMPTOMS.—The lips were very much swollen and covered with small pustules, both external and internal. There was a distinct slaty coloured line on the gums, breath slightly offensive, teeth were not discoloured and were not loose, slight dribbling of saliva. The pads of all four feet were very tender and "peeling," and the interdigital spaces were reddened and inflamed and a greasy exudate present. On grass the dog could walk fairly well, but on hard ground it was like the proverbial cat on hot bricks.

DIAGNOSIS.—In view of the gum discoloration, lead poisoning was suspected, and after a lot of questioning, the owner stated that two weeks previously he had been applying "what he thought was a lead ointment" to a cyst on the left hind leg, and that the dog had licked it off as fast as he put it on. On asking to see the ointment, I found that it was the ordinary mercuric ointment (or troopers' ointment).

TREATMENT.—This consisted in bathing the mouth with pot. chlor. solution, the feet with Caporit (Bayer) lotion, and then drying and dusting with a cooling dusting powder, and the internal administration of potassium iodide in 1-grain doses three times a day.

CUTANEOUS LEISHMANIASIS IN A DOG.

BY V. R. PHADKE, G.B.V.C., AND KHAN SAHIB A. I. SHAIKH, G.B.V.C.,
Professors in the Bombay Veterinary College.

HISTORY.—A brindled mongrel bull terrier pup (slut) about nine months old, with ulcers on the muzzle, upper lip, on both sides of the nostril, at the root of the right ear and inside the left ear, left hind paw and on the sternum, was sent to the Bai Sakarbai Dinshaw Petit Hospital for Animals on the 28th of April, 1924, for treatment and clinical demonstration by Dr. Row.

The pup was brought down to Bombay from Peshawar (N.W.F.) some six weeks prior to the admission in the above hospital (by Captain V. S. Pandit, I.M.S.). Dr. Row, on microscopical examina-

tion of stained slides made from the material scraped from the edges of unhealthy looking ulcers on the ear, noted bodies similar in structure to Leishman Donovan bodies of Kala Azar or Delhi boil in human beings.

Similarly, scrapings taken from the edges of the ulcers on the

FIG. 1

ear were stained and examined by us with positive results, thus confirming his diagnosis (Dr. Row's).

STATE OF THE ANIMAL ON ADMISSION.—The pup was in fair condition, temperature normal, appetite good, and bowels were regular.

FIG. 2.

SYMPTOMS.—The animal had ulcerations in the following situations :

(i) At the root of the right ear (outside) there were three big ulcers varying from a four-anna piece to a rupee, with raised irregular edges, the bigger one having a pitted centre ; inside the same ear one small irregular healing ulcer of the size of a pea was present.

(ii) *Left Ear.*—Inside the left ear, on the posterior portion of the conchal cartilage, eight ulcers varying from the size of a pea to that of a two-anna piece, with raised and thickened edges with pitted centres, unhealthy in appearance, were observed.

(iii) *Face.*—Three small ulcers of the size of a lental with greyish deposit were seen on the inner canthus of the left eye. Right eye free.

(iv) *Muzzle* (upper lip).—Ulcers on both sides under the nostrils (on the right side triangular about $3\frac{1}{4}$ inches in length, and on the left oval, of the size of an eight-anna piece) were present.

(v) *Nostrils.*—Upper surface was ulcerated and presented an appearance of being covered by dark dried blood. The edge of the wing of the left nostril inside was inflamed and thickened. The edges of the upper lip on both sides were denuded of skin and having a raw bleeding surface.

(vi) *Lower Lip.*—In the centre it had three small ulcers varying from a lental to a two-anna piece with raised surface.

(vii) *Left Hind Leg.*—On the left hind paw, just above the middle toe, one circular ulcer with very raw surface and slightly thickened edges (size eight-anna piece) was present.

One ulcer of the size of a pea on the right fore leg just above the carpal joint in front was seen.

(viii) *Sternum.*—One unhealthy looking ulcer about the size of an eight-anna piece in the centre of the sternum was present.

Ecto-Parasites.—No ecto-parasites of any description were found on the animal's body even after a careful and prolonged search.

Bacteriological Examination.—Preparations were made from scraped material obtained from the thickened edges of the unhealthy looking ulcers (with a scalpel), and also from small droplets of blood that oozed out and were stained by Leishman's and examined.

Numerous bodies were present in the preparation made from the scraped material, but very few in the blood smears. These bodies were oval in appearance. The nucleus was situated along the longer border and the rod-like micro-nucleus by its side. These bodies were scattered singly throughout the whole smear, and also in larger numbers in the cytoplasm of some of the large nucleated cells.

On the 29th of April, 1924, a photograph of the pup was taken in different positions, as shown in Figs. 1 and 2.

Treatment.—The animal remained under treatment from the 29th of April to the 31st of August, 1924, when it was discharged as cured.

The patient was put on a course of the following mixture :—

℞ Liq. arsenicalis .. .. ♏ x.
Vin. ipecac. .. .. .. ♏ xv.
Pot. citras. .. .. .. grs. x.
Mag. sulph. .. .. .. ℥i.
Aquæ ad. .. .. .. ℥ii.
M. ft. mixture.
Sig. Half twice daily.

Fig. 3.—Photograph after Cure.

All the sores were touched with acetic acid, followed by the application of the following liniment :—

℞ Acid carbol.
Chloral hydras.
Tr. iodi.—a.a. .. .. .. ℥i.
M. ft. liniment.
Sig. To be applied as directed.

On the 5th of May the above mixture was discontinued, and as the temperature of the animal began to rise (103°), febrifuge mixture was given. On the 7th of May, 1924, temperature rose to 104°, and then began to drop. On the 10th of May it was 101·6°.

On the 11th of May, 1924, the liquor arsenicalis mixture was again continued till the 19th of May, on which date the animal was put on a course of the following :—

℞ Mercuric cyanid. .. .. gr. ½
Pul. zingberis .. .. .. grs. 2
Ext. gent. .. .. .. q.s.
M. ft. pill.
Sig. To be given once a day in milk.

XUM

External treatment remained the same, except on occasions the ulcers were treated with liq. hydrogen peroxide prior to the application of the liniment.

The ulcers seemed to be more healthy looking, but there was very little tendency on their part to heal. From the 10th to the 30th of May temperature of the animal ranged between 101·6° to 102·8°, and the animal was put on a febrifuge, and the mercuric pill treatment was discontinued. The fæces remained loose and the animal was off his feed for a few days and had to be put on Dover's powder, salol and bismuth a.a., grs. x., half twice daily, and also the febrifuge mixture.

From the 2nd of June, 1924, a change in the dressing was effected, ferri arsenas being used as a dusting powder for the wounds and ulcers in various situations. In a few days' time this powder seemed to have a better effect and was continued till the end. But the ulcers were being touched sometimes with liq. hydrogen peroxide, silver nitrate and tr. of iodine.

The ulcers began to heal—the process of healing being retarded by the animal attempting to scratch with his paws, although they were bandaged.

TRANSMISSION EXPERIMENT.—Two pariah dogs were obtained from the lethal chambers for the above experiment.

Dog. No. I (*a white slut*).—An area of the size of a two-anna piece was scarified inside the right ear, and infected material, obtained by scraping from the edges of the ulcers on the ear of the affected dog, was placed on the scarified area and allowed to dry (30th of April, 1924).

Dog No. II (*black and brown dog*).—Two areas of the size of a two-anna piece inside the right ear, and one similar area on the inside of the left ear, were scarified, and the infected material placed and rubbed on these areas and allowed to dry (30th of April, 1924).

The infected material was subjected to microscopical examination, prior to placing the same on the scarified areas, and numerous oval bodies were found to be present in the stained preparations examined. While under observation these two pariah pups developed a cutaneous form of distemper and had to be treated. Within the course of a few days the scarified areas healed up completely, leaving no appreciable mark of the operation.

On the 13th of June, 1924, at the seat of the inoculation inside the right ear of dog No. I, the surface was noticed to be slightly red and raised (forty-four days after the inoculation). Three days after, definite nodule formation of the size of a two-anna piece well raised

above the surface took place, and this began to present an ulcerated appearance on the 18th of June, 1924.

After scarifying the edges of the ulcerated nodule, smears were made from the oozed out serum and stained by Leishman's, and on microscopical examination similar bodies were found to be present in the preparations. On the 18th of July, 1924, this ulcer began to heal, the surface being covered by a greyish scab. On the 28th of July, 1924, it healed up completely, but the next day, as a result of a scratching by the animal, the thin scab came off, leaving a sore surface. After a few days scab formation again took place. On the 5th of September, 1924, smears were made from the scabbing ulcer and examined with negative results. At this stage the thickening at the edges of the ulcers had almost disappeared, and on the 10th of September, 1924, it was found to have completely healed up.

FIG. 4.—Photograph of the Animal showing Ulcer in his Right Ear.

On the 15th of September, 1924, a small red nodule of the size of a lental appeared again on the same spot. Smears were made from the same, but were examined with negative results. This nodule became smaller and smaller and disappeared completely on the 23rd of September, 1924. The animal was kept under observation till the 17th of November, 1924, when it was eventually destroyed.

Dog No. II treated similarly, after forty-seven days, showed slight thickening and redness inside the left ear on the 17th of June, 1924. Two days after a nodule of the size of a pea, slightly raised above the surface, appeared at the seat of inoculation, but in the right ear only slight thickening was noticeable.

Preparations made from the nodule were stained by Leishman's and examined with positive results. In a few days' time the nodule in the left ear increased in size and started ulcerating, and on the 21st of July, 1924, the ulcer covered an area of the size of a two-anna piece.

In the case of the right ear, however, nodule formation was delayed

till the 22nd of July, 1924, when two small nodules appeared at the seat of inoculation (one in each area).

These began to ulcerate on the 25th of July, 1924. On the 5th of September, 1924, the ulcerating nodule in the left ear became bigger (of the size of a four-anna piece), with raised edges, and was covered partly with a greyish scab. Two ulcers similar in size and appearance were seen in the right ear also. These ulcers healed up completely on the 12th of October, 1924.

The animal remained under observation till the 17th of November, 1924, when it was destroyed.

It is interesting to note that there was no tendency on the part of the infection to spread, but it remained localised in the scarified and inoculated areas in both the ears, no fresh nodules being observed even near about the inoculated areas.

The animals gained weight, and at the time they were destroyed were nearly double the size when first inoculated.

Naturally infected dogs have been found in the Mediterranean countries (Algiers, Greece, etc.) where Leishmaniasis in infants is very common, and Basile has claimed that the disease is transmitted by fleas from dog to dog. Wenyon, however, reported to have failed in infecting dogs with fleas previously fed on dogs infected with canine Leishmaniasis, and suspected other agencies.

It has been suggested that the dogs of India where Kala Azar prevails may be immune, since no infected dogs have been found in Kala Azar districts, but the fact that the clinical case brought by Captain Pandit to Bombay from Peshawar (N.W.F.), where cutaneous Leishmaniasis (Delhi boil or frontier sore) does exist, goes to prove that cutaneous Leishmaniasis in dogs is also prevalent in India. Its relationship with the human disease is no doubt obscure.

In Bombay a case of canine Leishmaniasis has been recorded in the *Indian Medical Journal*, December 1924, by Khan Bahadur C. R. Awari and Lieutenant-Colonel F. P. Mackie, I.M.S., of the Bombay Bacteriological Laboratory, in a pariah dog.

This year in the month of March Mr. Phadke and Mr. Hewlett, Principal Bombay Veterinary College, observed a similar case in a stray pariah dog in the College compound, with superficial sores on its ears. The clinical picture presented by the ulcers situated on the external surface of the ear was identical with the case under reference. Unfortunately the animal could not be caught, and this fact precluded the possibility of arriving at a positive diagnosis under microscope.

However, the incidence of this disease in dogs in India appears to have a negligible proportion. Probably the canine disease may be classed as a separate entity from human disease. The fact that

Bombay is not a centre of Kala Azar or Delhi boil goes to support the above contention.

From the evidence available from the attempted transmission of infection to the two experimental dogs from the clinical case sent for treatment, it appears quite reasonable to conclude that in pariah dogs cutaneous inoculation only sets up localised lesions which heal in due course without any treatment.

CASTRATION OF THE RABBIT.

By CHARLES A. SQUAIR, M.R.C.V.S.,
Reigate.

It may not be often that a qualified veterinary surgeon is called in professionally to castrate a rabbit, but that it does occur as one of the items of daily practice came home to me as a fact a few days ago when I was asked by a client to operate on a certain number of Angora rabbits. As I had never done the operation before I was puzzled as to how to get hold of the testicles, for I found that the patient had the power of withdrawing them until they were quite out of touch. Upon referring to Professor Hobday's book on *Castration and Ovariotomy*, I found the statement that the rabbit had this power when frightened or otherwise aroused, and the method I adopted was as follows :—

Upon the assistant taking up the rabbit I attempted to seize hold as quickly as possible of the testicle, but finding it had eluded me I asked the assistant to give him a swing in the air just to temporarily upset his equilibrium. I found that then for a few seconds the testicles had descended, and seated on a chair with helper opposite on a chair, I readily performed the operation. I adopted the pulling method (as the tissues are very tender, and then there is no hæmorrhage), when once the organ was let out of the scrotum, in much the same manner as one would do a cat, and in no instance did the animals give any anxiety afterwards.

A CASE OF SWINE ERYSIPELAS TRANSMITTED TO MAN.

By R. J. STOW, M.R.C.V.S.,
Redhill.

On the 23rd of September of this year I was called in professionally to see three pigs, all of whom were very constipated and without appetite. One had a temperature of 106·5°, and the other 104·5°. Two others in the same sty were normal, as also about fifteen others in close proximity. On the 24th the temperature of one pig was

106°, and the other 106·5°. All the others were normal. On the 26th one was dead, and a third one was affected in an adjoining sty, with a temperature of 105°. The *post-mortem* on the dead pig showed slight pneumonia and inflammation of the stomach and intestines. In making this I cut my left thumb. After washing it in disinfectant I painted it in tincture of iodine. On the evening of the 29th I noticed a slight inflammation around the cut, which I bound up with lint soaked in 2½ per cent. carbolic, and covered with rubber finger-stall. Everything appeared to be going on all right till the 3rd of October, when the thumb became tender and swollen and very hot around the cut, which had now healed. On the 7th the whole thumb was very tender and swollen, so much so that on the 8th I thought it wise to seek medical advice. My doctor suggested painting the part with 2 per cent. picric acid in surgical spirit, and covering up around the lesion with collodion. I also covered up the part with wool and a bandage. On October 12th the thumb had greatly improved in appearance and was not painful. In the afternoon I played a round of golf, with the result that at night the thumb was swollen again and very painful, throbbing especially round the nail. Since then I have had very little pain or swelling, but the lesions keep changing their position, working more up towards the hand. At the present time I have a patch of inflammation running from the first finger for 2 inches across the back of the hand, and another small round spot around the ball of the thumb. The thumb appears quite normal, but feels slightly painful on pressure. I have not at any time had any pain in the lymphatic glands of the arm.

Translation

TUBERCULOSIS OF THE NERVE CENTRES IN THE OX AND PIG.

By A. CHRÉTIEN.*

Tuberculous lesions of the nerve centres in the domestic animals are relatively rare and most often affect the meninges. We record hereafter two slightly different cases recently observed in meat at the Paris Abattoir.

I. One of the cases relates to the brain of an ox seized by our confrère M. Bouffanais.

This brain, which was part of a lot of frozen brains dispatched from a foreign country, presents in the left hemisphere, in the thick-

* *Rec. de Méd. Vét.*, 1925.

ness of the parietal lobe, a series of confluent tubercles of a greyish yellow tint, of hard consistence, calcareously infiltrated, macroscopically comparable to the usual tuberculous glands. The meninges at this place seem hardly to participate in the infection.

Macroscopic diagnosis, which presents no difficulty, is confirmed by microscopical examination, and on preparations stained by Ziehl's method, tubercle bacilli are very numerous, contrary to what one usually observes in tuberculous lesions of the ox. Histological examination shows everywhere a series of tubercles whose central caseous portion is calcareously infiltrated in places; the periphery of the tubercles, formed of epithelioid cells, encloses a large number of giant cells. Tubercle bacilli are plentiful in all the areas of the tubercles.

A few such similar lesions have already been observed and described in the ox by M. Moussu and M. Besnoit.

II. The other case concerns a pig sent in without head and seized by M. Gély for generalised glandular tuberculosis.

The majority of the lymphatic glands present classic tuberculous lesions. As regards the central nervous system the spinal marrow shows very discrete lesions of meningitis; in the lumbar region for a length of some centimetres the marrow is hypertrophied, indurated, of uniform greyish tint, and infiltrated with fine calcareous granulations, the roots of the nerves being similarly affected. The neighbouring vertebræ are normal.

Microscopic preparations give no evidence of Koch's bacilli, but inoculation positive to the guinea-pig shows the bacillary nature of the lesion.

On histological examination one observes quite a series of small grey tubercles, somewhat calcified at the centre, but without the least trace of caseation. Giant cells are excessively rare. Almost all the substance of the marrow is invaded with tubercles of this nature, and on section only a few zones of white substance seemed to have preserved their integrity. Although all tissues are susceptible to invasion by tuberculous infection, in these cases there is a localisation of exceptional rarity capable of provoking in the living animal diverse locomotor troubles difficult to diagnose.

Abstracts of Current Literature

Meat Poisoning in Prussia.—*The Lancet.*

An interesting article * by Professor O. Lentz supplies a good deal of information upon outbreaks of meat poisoning occurring in Prussia between 1907 and 1924, and incidentally throws some sidelights upon the shortage of meat in that country during and after the war. By virtue of an enactment of August 1905, outbreaks of meat poisoning in Prussia have to be reported by the district medical officers to the appropriate Government department. These reports, although showing some omissions, are available for a survey of the causes and course of these outbreaks. Lentz gives the following table :—

Year.	No. of Outbreaks.	Approx. Total No. of Cases.	Deaths reported.	Year.	No. of Outbreaks.	Approx. Total No. of Cases.	Deaths reported.
1907	10	294	1	1916	7	906	1
1908	5	283	3	1917	8	1,119	19
1909	4	111	3	1918	2	433	20
1910	6	395	2	1919	8	530	9
1911	4	156	2	1920	18	1,016	12
1912	8	667	6	1921	34	1,934	17
1913	8	928	3	1922	55	2,633	18
1914	5	69	6	1923	36	2,095	8
1915	—	—	—	1924*	7	52	4

* First four months.

While the majority of outbreaks are ascribed to infection with bacilli of the paratyphoid-enteritidis group, eight are credited to *Bacillus proteus* and twenty-four as examples of botulism. In only one of the latter, however, was *B. botulinus* isolated with certainty, while in two others anaerobes of the type of this organism were present.

The causation of this serious rise in the number of outbreaks is discussed by Lentz. While admittedly some of the outbreaks were associated with the use of food which was perfectly sound, in the majority the meat consumed was derived from animals definitely diseased. In pre-war times such food was very rarely passed and allowed to be eaten, but in consequence of the great shortage of meat during the war and in the after-war period, especially during the years of inflation, more and more necessity-slaughtered meat and meat which eluded inspection altogether was sold for human food. According to Lentz, this assumed very large proportions, and he

* O. Lentz: Ueber Fleischvergiftungen, 1924, *Zeitschr. f. Hygiene und Infekt.*, ciii. Part 2, p. 321.

quotes various reports as bearing out this contention. For example, in the Berlin Kadaververwertungsanstalt during the year 1914, 4,500,000 kilos of animal carcasses and decomposed meat was dealt with, while in 1923 the corresponding amount was only 730,000 kilos. The natural suggestion that during this later period proportionately fewer animals departed this life is countered by the statement that during and after the war, especially during the inflation time in Berlin, a great deal more sausage meat was actually consumed.

Negligent inspection must, in Professor Lentz's view, bear a large part of the blame for this increase in meat poisoning. In many cases, in spite of offensive septic and other conditions in the animals, the meat had been stamped as fit for food. He gives an instance of a veterinary inspector passing the meat of a cow, in spite of the presence of a much enlarged spleen and small hæmorrhages upon the serous membranes. Two of the slaughtermen concerned subsequently fell ill with anthrax and one died. In spite of repeated reminders from the central authority, bacteriological investigations of suspicious cases are seldom resorted to, and usually the guide utilised is merely the colour and consistency of the flesh. Another instance given is one in which the meat inspector passed meat as fit for use which was definitely suspicious, the only test employed being that he and his family ate a piece of it, his only proviso that it was to be well cooked. This meat caused an outbreak of meat poisoning, due to *B. enteritidis*, involving sixty-two persons. In only two instances from the many reports is it mentioned that severe offences against the regulations have resulted in the removal of the meat inspector from his office. Legal proceedings against offenders have been quite ineffectual. Lentz presses strongly the need for routine bacteriological examination of all necessity-slaughtered animals before being passed for human consumption. These facts suggest the reflection that the relaxation of a rigid system of meat inspection amongst a people accustomed to be guided by rule is likely to be fraught with greater risk of disaster than a limited inexact system of inspection amongst a community forced to exercise discrimination of their own.

Leiper, R. T., M.D., D.Sc., F.R.S.—The Public Health Significance of Recent Advances in Helminthology.—*Journal of State Medicine.*

THE intense activity with which research has been pursued during recent years has resulted in knowledge which, if properly applied, should ensure the control and eventual eradication of the infestations of man with parasitic worms. From the Public Health standpoint the significant facts are :—

(*a*) *The parasitic worms do not multiply within the human body.*

Although they produce innumerable eggs or embryos, these are unable to develop within the body into a new generation of adults.

(*b*) *The eggs or embryos must first leave the human body which harbours the parents.* The great majority leave in the fæces. In one case the eggs appear in the sputum, but as the bulk of the sputum is swallowed they also are mainly passed in the fæces. In another instance the eggs are voided in the urine. The embryos laid by the intestinal worm *Strongyloides* are passed in the fæces, but those of the *Filaria* worms which circulate in the blood can only leave if sucked up by a biting insect. The guineaworm is peculiar, for the female discharges her young through a breach she makes in the skin, in the hope that they may reach water.

(*c*) *The eggs or embryos are not infective to man at the moment of leaving the human body.* A period of delay is essential before this non-infective stage can become infective (save in those cases in which man can also become infected with the larvæ).

(*d*) *Developmental changes occur during the period of delay outside the body, and are essential to the formation of the "infective" stage.* These changes in some instances result only in individual alterations, in others there is an asexual multiplication with the formation of numerous infective forms from the single embryo.

(*e*) The environmental conditions requisite for the developmental changes outside the body vary with different species. It is rare for the extra-corporeal phase to be able to withstand drying, and it may be accepted that helminth infections are not blown about in dust. Owing to the relatively long period of delay necessary for development the mechanical transmission of helminth eggs by flies may be ignored as of no practical significance. In some instances (as in *Oxyuris*, *Ascaris*, *Trichocephalus*), moisture is sufficient; in others water is necessary, in others still the egg must first enter or the embryo pierce some other animal, usually invertebrate (arthropod or mollusc), more rarely another mammal. There are cases where the developmental stage leaves this "intermediate host," and secretes itself in another host, in the tissues of which it is then swallowed by man.

(*f*) The infective stage enters the body in most cases by the mouth in food or as contaminations of food; but in certain instances the infective stage actively pierces the skin: In hookworm and *Stringyloides* from a moist surface, in *Bilharzia* during immersion in infected water. Drinking water (except as a vehicle for some encysted flukes, and for *Cyclops* the intermediate host of guinea-worm) may be regarded as an unusual medium by which helminth infections gain access to the human body. If considered safe bacterio-

logically, it may be regarded as also safe from the helminthological standpoint.

(*g*) After entry, the infective stage has, in many species, to undertake an extensive pilgrimage within the body; in some cases through the bloodstream, in others through the tissues or serous spaces, before it can successfully attain to sexual maturity in its normal *habitat*. These migrating forms may cause serious damage to the tissues they are traversing (e.g. pneumonic changes follow upon the migration of ascaris larvæ through the lung).

(*h*) Few of the parasitic worms are specific to man (e.g. *Ancylostoma duodenale*). It more frequently happens that the same species is also prevalent in one or other of the domesticated animals. These additional "reservoir" hosts may play an important rôle in spreading infection (e.g. *Schistoma japonicum*, the cause of bilharziosis in man, in the Far East occurs in dogs, cats, and cattle in the endemic regions).

The spread of helminth infection can be controlled by breaking the life cycle of certain vulnerable points:—

(1) The adults may be killed by anthelmintics while within the human body. (This applies to intestinal forms.)

(2) The eggs or embryos may be killed while within human tissue by therapeutic agents. (This applies so far only to the Bilharzia eggs, but a similar remedy against the Filaria embryos is being eagerly sought.)

(3) The eggs and embryos, when passed in the body discharges, may be "side-tracked" by "conservation" measures, or in many cases simply by lack of access to water necessary for the hatching of the eggs.

(4) Re-entry of the infective stage may be prevented by: (*a*) Avoidance of exposure to infective soil (e.g. by the use of boots and clothing to protect the skin in the case of hookworm); (*b*) avoidance of open bathing in canals and ponds liable to be contaminated (e.g. Bilharzia); (*c*) avoidance of raw unfiltered water for drinking (infection by free eggs or embryos in such water can however occur but seldom, as these rapidly sink to the bottom owing to their weight); (*d*) the cooking of fish, meat, and shell-fish (including in this term fresh-water prawns and crabs), and of vegetables upon which eggs may have been deposited (e.g. *Ascaris*, *Trichocephelus*) or infective larvæ become encysted (e.g. *Fasciolopsis buski*). This involves the avoidance of salads and of Oriental sauces of which uncooked fish may be an ingredient.

(5) Where an intermediate host is essential, a knowledge of its bionomics or susceptibilities may suggest means by which it can be eradicated, e.g.: (*a*) The use of chemicals such as copper sulphate

which have a specific lethal action even in high dilution against fresh-water molluscs; the introduction of ducks and the sudden exposure of the molluscs to drying, as has been suggested for the control of bilharziosis in Egypt; (*b*) the reduction of numbers by hand collection, as has been tried against the *Melania* molluscs responsible for the spread of lung-fluke in Asia; (*c*) the application of anti-mosquito measures for the control of filariasis.

To be in a position to utilise the knowledge which we now possess concerning the parasitic worms, the Medical Officer of Health should have some acquaintance with the morphology of the principal helminths of man; their normal *habitat*, and their geographical distribution; the manner in which their progeny leave the body; the method of collecting samples, and the means by which the various species can be identified from microscopical characters of the eggs and embryos; the after history of these eggs or embryos outside the body; the intermediate hosts which in certain cases are essential; the exact rôle that these intermediate hosts play in procuring the re-entry of the parasite into the human body; the particular route of entry of the infective stage; the local reactions set up by the invading form at the seat of entry, and in its course of migration through the body previous to attaining its normal *habitat* as an adult; the value of the various anthelmintics in the treatment not so much of those suffering from helminth disease as of those healthy persons with slight infections who act as carriers and disseminators.

Mackenzie, K., M.A., and Marshall, F., Sc.D., F.R.S.—On the Presence of Supernumerary Mammary Glands in Cows and on their Functional Activity.—*The Journal of Agricultural Science*, 1925.

BATESON in his book on variation (*Materials for the Study of Variation*, London 1924) has the following: "Normally the cow has four teats of about equal size. Not unfrequently there are six teats, of which four are large, and may be said in the usual parlance to be the normal ones, and two are small, and placed posteriorly to the others. . . . Commonly these extra teats give no milk, but in many cases they have been known to do so. Their size and position vary greatly; sometimes they are placed near the other teats . . . but I have seen them very high up, almost in the fold between the udder and the thighs. Very frequently, however, there is only one extra teat, making five in all, such an extra teat being, so far as I know, always on one side of the udder."

Burchhard (*Anat. Hefte*) and Henneberg (*ibid*) state that 38 per cent. of cows possess additional teats. Both these authors say that

they vary from one to four in number, and are usually situated behind the normal teats and occasionally between them. Burchhard also records supernumerary teats on the scrotum of bulls. Henneberg concludes that the accessory glands lower the milk production by causing the other glands to secrete less than normal.

[The converse of this has been believed to be true by dairymen and farmers hitherto. Supernumerary or small extra teats have been said to be desirable as indicating good milkers, and small teats on the purse of a bull are considered beneficial as evidence of a sire likely to beget good milking stock.—G.M.].

The present investigation was suggested by the observations and experiences of one of us (K.J.J.M.). It was found to be the custom in some countries, and more particularly in parts of Holland, to remove the extra teats from the calves. The glands, however, were apparently not removed, and these were sometimes seen or felt as slightly distended portions of the udder, which occasionally showed evidence of an inflammatory condition. In this country it is not the custom to remove the additional teats, but they are not milked from, and are commonly regarded as being functionless.

It was the purpose of this investigation to obtain evidence as to the frequency of occurrence of these additional structures, and secondly to ascertain whether the glands were functionally active.

Herds consisting of Shorthorns, half-bred Ayrshires, South Devons, North Devons, and Jerseys were examined, and out of 276 cows comprised in them 154, or rather more than half, had extra teats. In most of these cases there would be extra glands associated with the teats, each teat would have its separate gland, and if a teat was cut off the corresponding gland could not be drained by the remaining teats.

It occurred to the investigators that since the milk secreted by the extra or accessory glands is not drawn off in milking the constituents must be absorbed into the circulation, and that one might expect under such conditions to discover lactose in the urine. We therefore examined for lactose samples of the urine of all those cows on the University Farm which possessed extra glands.

The method adopted was that of Cole (*Lancet*, September 20, 1913). In cows with normal four teats no absorption of lactose took place, whilst in those with extra glands absorption of lactose from the udder into the circulation was found to be going on. The conclusions arrived at were :—

1. Rather over 50 per cent. of the cows in this country probably possess accessory or supernumerary teats, the number of which varies from one to three.

2. In many cases, at any rate, these teats are associated with additional glands.

3. These additional glands are frequently functional and secrete milk.

4. Ordinarily this milk is absorbed into the circulation, the lactose being secreted in the urine.

5. The presence of active accessory or supernumerary glands is an undesirable characteristic, since it involves the formation of milk which is not utilised.

Cameron, T. W. M., M.A., B.Sc., Ph.D., M.R.C.V.S.—Studies on Two New Genera and Some Little Known Species of the Nematode Family "Trichostrongylidæ."—*The Journal of Helminthology*, vol. i. pp. 71–96.

THIS paper describes six species of roundworms from various animals, domestic and wild.

Graphidium strigosum, a well-known stomach-worm of rabbits, and occasionally the cause of a fatal gastritis, is described and figured in greater detail than hitherto.

Mecistocirrus digitatus, a parasite resembling *Hæmonchus contortus*, and of some importance in ruminants and the pig in Asia, is described and its affinities discussed. It is shown that it is closely related to the stomach-worm of ruminants, differing from this species in the presence of long spicules in the male and in the absence of an asymmetrical bursæ lobule and a vulvular flap. It also appears to cause a parasitic gastritis, and it is suggested that it may be much more widespread than is generally supposed. It has been known since 1906, and has been reported from Malay States, Japan, Hongkong, and India. From the latter country it has been described by Sheather, who mistakenly considers it to be an unknown variety, under the heading of "a new nematode causing parasitic gastritis in calves."

Molinens felinens is the first species of this family reported from cats, in this case from *Felis yaguarundi* from South America. As is the case with so many feline parasites, this worm is probably not restricted to wild cats, but may be found in domesticated varieties also. It is a very slender form, about 5 mm. long, and can easily be overlooked.

The paper concludes with a valuable key to all the known genera of *Trichostrongyle* worms.

Review

History of the Great War based on Official Documents—Veterinary Services. Edited by MAJOR-GENERAL SIR LAYTON J. BLENKINSOP, K.C.B., D.S.O., and LIEUT.-COLONEL J. W. RAINEY, C.B.E. Published by His Majesty's Stationery Office. 21s.

This is the story of the way in which the Royal Army Veterinary Corps performed its part in the Great War, how it was organised on the experiences gained in the South African War, and how its organisation was developed, improved, and perfected to meet the unprecedented demands of a world war. It is a story of which the veterinary profession may well be proud. It is written in no boasting spirit: defects, mistakes, mismanagements are all frankly laid bare, but always the experiences were turned to profit. By the time the war ended the Veterinary Services were responsible for tasks undreamed of before, and in this volume all is recorded, making it a priceless storehouse for future guidance. The different theatres of the war are each described by the officers in charge, some of whom kept personal records which make interesting reading. Special chapters have been written on the Veterinary Service in connection with the Remount Commission, the duties of the veterinary officer at the Front, the construction and organisation of veterinary hospitals for horses, mules, and camels respectively, the ordinary and special surgery and surgical procedures, the transportation of animals across the seas, etc. The work runs to 775 pages, and is provided with a useful index. It is the most exhaustive account ever written of the work of the veterinary service in war. "The onerous duty of co-ordinating the various parts, and preparing the whole for publication," says Sir Layton Blenkinsop in his preface," has been performed by Lieut.-Colonel J. W. Rainey." We congratulate him on having produced an admirable record, and to him and to all those who have contributed to this monumental work we offer in the name of the profession our congratulations.

Personal

The Walley Memorial Scholarship for 1925 has been awarded to MR. HAROLD BURROW, of the Royal (Dick) Veterinary College, Edinburgh. This prize is awarded annually for competition between all the colleges in Great Britain and Ireland to the student who obtains the highest percentage of marks in the Third Professional Examination.

DR. W. J. HOWARTH, C.B.E., M.D., Medical Officer of Health for the City of London, has been selected by the Model Abattoir Society to give the Benjamin Ward Richardson Memorial Lecture. The meeting will be held on Friday, November 20th, at 5.30 p.m., in the Barnes Hall of the Royal Society of Medicine, and the title chosen by the lecturer is "The Slaughtering of Animals for Human Consumption: Observations on Methods Practised, and Desirability of Centralisation in Relation to Meat Hygiene." Members of the veterinary profession and students will be welcomed.

The numbers of lady students in our British colleges are gradually

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increasing, and Glasgow has now been invaded, as the Glasgow School has three lady entrants. In the Utrecht University Veterinary School, too, a lady student is in the classes of the first year, and this new innovation is being watched by the veterinary profession in Holland with great interest.

Congratulations to CAPTAIN GERALD WACHER, M.R.C.V.S., on the presentation which was recently made to him of the Medal of the Royal Society for the Prevention of Cruelty to Animals for his share in the rescue of a pony which had fallen down a deep pit in Impkins Wood, near Charing. The animal was imprisoned down the pit for fourteen hours, and the rescue party had to descend by the aid of long ladders lashed together. Owing to the bad air at the bottom they were only able to work for a short time without returning to the surface; and on account of the small space and the narrowness of the passage, it was only with the greatest difficulty that the pony was eventually pulled out.

Notice

THE Central Branch of the National Veterinary Medical Association are holding their Annual Dinner at the Trocadero at 7 p.m. on November 5th.

Major-General Sir John Moore, K.C.M.G., C.B., F.R.C.V.S., the newly elected President, will be in the chair, and a distinguished gathering is expected.

NOTICES

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